Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

February 7, 1899.

Vice-President, Dr. Joseph Collins, in the chair.

CASES OF PARTIAL SPINAL CORD INJURY.

Dr. Pearce Bailey presented two cases of this kind. The first was that of a man who on December 9, 1898, fell several feet, struck on his head, and became unconscious. On recovering consciousness he had tingling in both upper and lower extremities, but no disturbances of the bladder or rectum, and no real paralysis. He soon afterward left the hospital, but since then had been troubled with pain and stiffness in the neck, and with numbness in the extremities on the right side. Examination at the time of presentation showed anesthesia of the right forearm and right leg, more marked in the latter, and a difference in the palpebral fissures. The left pupil was the smaller and less responsive. No clonus, no paralysis and no twitchings were observed. A distinct prominence was seen in the cervical region, but the stiffness of the neck was rapidly diminishing. The case was looked upon as one of slight unilateral hemorrhage in the sixth cervical segment, with possibly some contusion.

The second case was that of a man, forty-three years of age, who had been squeezed between two heavy masses in an elevator shaft last November, and while thus caught the elevator had descended upon his head, and pressed him down to the floor. He had not regained consciousness until after reaching the hospital. He had then severe pain in the neck and tingling in the fingers, but had control over the bladder and rectum. Examination at the time of presentation showed anesthesia over the right deltoid region, and exaggeration of all the reflexes, but the right patellar reflex was not as lively as that on the left side and a left foot-clonus had been noted. The case was an incomplete type of Brown-Séquard's paralysis, In view of the forcible flexion of the neck it was almost certain that a hemorrhage into the cord had occurred.

Dr. Edward D. Fisher said that he had under observation a case in which his diagnosis had been meningeal hemorrhage, and possibly a slight hemorrhage into the spinal cord, but because of the history he could not feel that a very serious loss of substance in the spinal cord had occurred. The condition of the reflexes seemed to indicate the existence of some compression outside of the cord, either the result of hemorrhage or of slight dislocation of the vertebræ. Three months had elapsed since the accident, yet the boy was only just able to move around. Unless paralyses were associated with atrophy he did not feel sure of the existence of a large hemorrhage in the cord.

Dr. George W. Jacoby believed that cases of this kind were examples of subarachnoid hemorrhage, and that the diagnosis could be made by lumbar puncture. If pure fluid, free from admixture with blood, were drawn off, there was evidently no subarachnoid hemorrhage. He also thought that this procedure possessed some therapeutic value.

Dr. Bailey stated that the diagnosis in his cases had been based mainly on collateral evidence. The records of autopsies showed that extradural hemorrhages were extremely rare, except when associated with mutilation of the cord. In the examinations that he had made on spinal cords of several persons who had met violent death from general traumatisms, he had found small hemorrhages into the cord without any fracture of the vertebræ. This was particularly true of injuries in the cervical region.

Dr. W. M. Leszynsky presented a case of spinal cord lesion with anomalous symptoms. (See p. 231.)

DEMONSTRATION OF THE BRAIN OF A CASE OF SENSORY APHASIA.

Dr. Joseph Collins reported the case of a negress, who had sensory aphasia. The patient when first seen had hemiplegia, not entirely typical, and at intervals of eight months had had attacks of epilepsy. The most striking defects of spontaneous speech were amnesia of words, particularly of nouns, and paraphasia. She could usually tell her name, but not the names of members of her family or her residence. It was impossible for her to repeat the simplest sentence after it had been spoken. She could sing the airs of popular tunes, but could not get the words in right. She had lost the comprehension of written and printed words. After repeated examinations right lateral hemianopsia had been demonstrated. She had had a slight degree of word-deafness, which became more apparent the longer the case was studied; it was due apparently to functional word degradation. A lesion of the visual center and subjacent white substance was believed to be present. If she were asked to say "B," she might say "C" instead, and in other ways she presented many instructive contrasts in connection with her aphasia. She died about two months ago.

On post-mortem examination, the brain was extremely small, and the left hemisphere was distinctly smaller than the right. There were also three areas of softening in the left hemisphere—one involving the middle third of the ascending parietal convolution; a second area situated in the posterior end of the inferior parietal convolution, and involving the entire angular gyrus, except its inferior portion, and a third area in the posterior end of the supratemporal convolution. The first area of softening measured 3 cm. perpendicularly, and 1.6 cm. at the widest part. The second area was the largest, measuring in its antero-posterior diameter 2 cm., and 1.5 cm. vertically. The most conspicuous alteration of the superior temporal convolution was its shrinkage, so that, as compared with the other side, it looked like a ribbon. The pia was not adherent except at one portion on the left side, and over the posterior end of the fissure of Sylvius. When the pia was stripped from the fissure of Sylvius, the destruction of tissue in the areas referred to was seen to be greater than had appeared at first sight. The cuneus had remained intact. The base of the brain showed no abnormalities, except the relative smallness of the left hemisphere. The optic nerve and chiasm were normal, the blood vessels were not thickened, and there was no evidence of meningeal inflammation. The medulla oblongata and cerebellum were normal in appearance and relationship. These findings seemed to him exceedingly interesting in view of the clinical history and the diagnosis made during life. He expected, on dividing the brain, to find that the optic radiations had been severed, thus giving rise to the homonymous hemianopsia.

Dr. Fisher expressed his surprise that in a case lasting only three years there should have been such a marked difference in the two hemispheres.

Dr. William Hirsch read a paper on the relation of infantile spinal paralysis to spinal diseases of later life. (To be published in this journal.)

Dr. Edward D. Fisher agreed with the reader of the paper that the case was not one of amyotrophic lateral sclerosis. The point had been very clearly brought out that many cases formerly diagnosticated as systemic diseases were really various forms of diffuse myelitis. He said that many cases of poliomyelitis were met with which extended over a long period, and yet they did not give rise to the condition found in the case just reported; hence, the direct relation of the inflammatory condition to the original poliomyelitis could not be definitely established. The theory of Strümpell that a virus could remain latent for many years and then exert its influence seemed to him very far-fetched. He would like to ask whether there was any direct connection between the bulbar symptoms and the spinal symptoms which were present primarily. The clinical picture of poliomyelitis was almost perfect, so that no other diagnosis could be expected under the circumstances.

Dr. Leszynsky said that he had had under observation for eight years a lady now of forty-eight years, who during her childhood had had a poliomyelitis. This had left her with an incompletely paralyzed limb. Some years later, she had developed loss of power in both lower extremities, and when first seen by him some years ago all of the signs of a progressive chronic poliomyelitis in both lower extremities were present. The disease had progressed, although very slowly, since that time. This seemed to indicate the possibility of a direct relationship between the old lesion and the later one, but this was the only case of the kind that he had ever encountered.

Dr. George W. Jacoby asked if cases of encephalitis occurred frequently or at all in later life after cerebral hemorrhage in childhood. He had seen one such case, in which an encephalitis occurred in an adult who had had a cerebral hemorrhage in childhood. There seemed to be an analogy between this and the case reported in the paper.

Dr. Joseph Collins did not think that there was any relation between the poliomeylitis and the diffuse myelitis occurring later in life in Dr. Hirsch's case. He could not imagine how the previous scar could have anything to do with starting the later inflammation from which the patient died. According to clinical experience, with the bare exception of keloid, scars were not the starting-point of such pathological processes. The whole thing seemed to him a mere coincidence. In spite of the great care exercised in the examination he had not been convinced that any definite relation between the two diseases had been demonstrated. It was naturally very gratifying to him that Dr. Hirsch's study had confirmed his own observations regarding the allocation of function of the posterior group of cells in the eighth dorsal segment. The explanation offered by the reader of the paper for the absence of changes in the Rolandic cortex also seemed very plausible and trustworthy. He certainly agreed with Dr. Fisher that it was ridiculous to suppose that a scar could lock up a poison for half a lifetime, and then allow it to come out and produce disease.

Dr. Hirsch closed the discussion. Regarding the connection of the bulbar symptoms and the spinal cord symptoms, and the relation between the infantile spinal paralysis and the later disease, he said that he had quoted theories largely, but it seemed to be an actual fact that the pathologic process started from where the so-called scar of the poliomyelitis had been. This had been proved both by the clinical symptoms and the pathological findings. The fan-like proliferation of connective tissue into the pyramidal tract accounted for the rigidity of the limbs at the beginning of the later disease. The order of development of the symptoms seemed to point to the origin of the later disease from the left anterior horn. Strümpell had compared the "scar" to a tubercular focus—evidently not an absurd comparison. He had found the report of a case of infantile poliomyelitis in which encephalitis occurred in later life.

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63. FO FILFÄLDE AF LANDRY'S PARALYSE (Two Cases of Landry's Paralysis). A. Schultz (Nordsk. Mag. f. Laegevidensk, 59, 1898, No. 6).

One of the cases here reported is of interest in that it presented all of the clinical features of an acute ascending neuritis, resulting in death by paralysis of respiration at the end of two weeks, and yet the microscopical findings, by modern methods, were entirely negative. The second case followed a multiple neuritis in a chronic alcoholic patient. Here there was a diffuse parenchymatous myelitis and a parenchymatous degeneration of the nerves. JELLIFFE.